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Epilepsy—doctor's dilemma, lawyer's delight? Medico-legal consequences of practising in the field of epilepsy report of an International League Against Epilepsy British Branch meeting—Edinburgh, April 2000

TIM BETTS

Birmingham University Seizure Clinic, Queen Elizabeth Psychiatric Hospital, Birmingham B15 2QZ, UK. E-mail: t.a.betts@bham.ac.uk

Six cases are described where the medical management of a person's epilepsy was brought under legal scrutiny. Lessons learnt from this educational exercise include improving doctor patient communication, the function of a Coroner's Court, when is misdiagnosis negligent, the vagaries of expert witnesses, should failure to diagnose a tumour be blamed on the physician or the service when facilities are inadequate, is failure to recognise a rare drug interaction, failure to warn against an interaction, or failure to take a proper history, negligent? The conference also examined the legal ramifications of the nurse/doctor relationship in epilepsy care, the place of epilepsy guidelines and, due to its interactive nature, reflected on the audience's epilepsy knowledge, which, in places seemed significantly deficient. It was a gripping educational exercise.

INTRODUCTION

The other day, in clinic, I realized I had made a mistake. I had been asked to review a man last seen by me 2 years before. At that time he had presented for a second opinion about diagnosis. He had a long history of recurrent affective disorder—often with fairly rapid onset and offset—almost invariably of a depressive nature (he had been 'high' occasionally). His astute General Practitioner (GP) wondered whether these short lived but intense, depressive spells might have an epileptic basis to them, particularly because they were often accompanied by an olfactory experience. He was already taking carbamazepine as a mood stabilizing drug.

Now, ictal depression does occur and indeed I have described it[1]: but in over 30 years practice of epilepsy I have seen perhaps only half a dozen cases. In all of them the drop into depression was extremely rapid, the depressive experience most closely resembled a depressive stupor and recovery was swift, after no more than an hour or so (although one patient managed to cut his throat in that hour)[1].

There was no doubt that the man in front of me did have recurrent episodes of depression of psychotic intensity: but they were often of 24 hours duration or longer and of relatively slow onset and offset: many seem related to his unhappy life experiences. His olfactory experiences seemed affect laden and mood congruent and were, I thought, as many 'temporal lobe symptoms' are[2], related to his psychiatric disorder although a little sharper and clearer than usual in such cases. His electroencephalogram was reported as showing mild intermittent non-diagnostic slowing over the left temporal area (a not uncommon finding in people with affective symptoms) and a high quality MRI was reported as normal.

On taking what I thought was a thorough history, I did not get the flavour of temporal lobe epilepsy and said so—the man departed much relieved that he had not acquired a diagnosis which would have made further changes in his life.

Two years later he was back in front of me. All had gone well (or at least as badly as usual) until 3 months before this present interview when carbamazepine had been withdrawn and lithium substituted—fairly abruptly. Shortly thereafter he had two witnessed tonic–clonic seizures both preceded by his olfactory experience. He was admitted to hospital and put on to 1000 mgs a day of sodium valproate. Since then he had had no further seizures and no further olfactory experiences or depressive episodes. An electroencephalogram now showed significant focal slowing over his left temporal lobe and an MRI scan (performed on

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the same machine and with the same reporter) now showed clear evidence of left hippocampal atrophy.

Probably, therefore, the experiences that I had categorized 2 years before as non-epileptic were actually epileptic in nature.

I do not think I had been swayed too much by my usual desire not to make a diagnosis of epilepsy unless clinically sure (and by my awareness of the overdiagnosis of temporal lobe epilepsy by my psychiatric colleagues in patients with 'soft' neurological symptoms). My clinical experience and reading of the literature, however, had been misleading: and this present patient's experiences must be added to and modify the clinical data base stored in my cortex. A salutary experience!

But one, I reflected, that might have been even more salutary. Suppose the man had died in his seizure or, whilst driving, had killed others? What would my position have been then? In a court of law, could colleagues have been found to defend my actions: or would one have been found to label my diagnosis as negligent? A missed diagnosis is not necessarily malpractice but leaves the perpetrator feeling uneasy.

There were, of course, other explanations for my patient's seizures—sudden withdrawal of carbamazepine, institution of lithium in someone prone to seizures: even the loss of his affective symptoms might be ascribed either to the tonic–clonic seizures themselves or to the mood stabilizing effects of valproate. It would have been an interesting few days in court, with themes expounded and dismissed (plus, no doubt, a scornful neurologist or two pontificating on the dangers of letting psychiatrists loose in the world of the brain). Interesting for the spectators, but stressful for the defendant. No one likes to have their actions and decisions tested in a court of law, no matter how well intentioned they seemed to be at the time.

I reflected on this experience as I began to write up the results of an interesting educational experiment the British Branch of the International League Against Epilepsy had carried out in Edinburgh a few months previously when we had examined some epilepsy related cases, that had either come to legal notice or had come before the courts, to try to learn from them.

Years ago, when I started my medical training, the management of epilepsy was cheap, non-contentious and easy. The diagnosis was made on clinical grounds through experience. Investigation was a skull X-ray and an EEG (or not): patients were then given either phenobarbitone or phenytoin (or both) and sent back to their GP. Some got better, some did not. Some died, some became wheelchair bound through ataxia, some became brittle boned, a few went mad, some had deformed offspring. That was the way things were with epilepsy. No one was to blame: it was fate. No need to blame the doctor, no need to sue: he had done his best.

Now epilepsy management is expensive. Investigation is complex and often not available or has a long waiting list although the diagnosis is still a clinical one. Treatment has advanced and there is a bewildering variety of drugs to choose from, some much more costly than others and all claiming to have specific advantages (but in a Health Service run by managers who 'know the price of everything but the value of nothing' cost has more clout than efficacy). Patients may remain in the hospital clinic now, rather than going back to their GP, and much of their management may be in the hands of a specialist nurse. There is much therapeutic optimism, but patients still fail to get better, may still die, still suffer side effects (often now because of interactions between medicines), may still go mad and may still have damaged children.

What has changed is patient and public attitudes to medical care and responsibility. If someone does not get better, suffers a medical mishap, or dies then someone must be held to account or sued for damages. This change in attitude had taken doctors by surprise and unprepared and muttering darkly about lawyers fostering a blame culture for their own ends and politicians stirring up anti doctor feelings to try to wrest control from the profession and to conceal the huge gaps in health care provision that have become the norm in our third world Health Service. This change has effected epilepsy management and an increasing number of epilepsy cases are going through the courts and an increasing number of clinicians are receiving solicitors letters alleging that poor or sub-standard seizure management had led to some mishap to their client and are seeking compensation. The situation has been complicated by the publication of guidelines for several aspects of epilepsy care with the implication that there is now a standard practice, deviation from which may be held to be incompetent.

All clinicians who manage epilepsy need to think about their standard of clinical practice and whether it would be harshly judged in a court of law or would be defensible if challenged. They need to think, too, about the actions of their team and where responsibility lies in the interaction between GP and consultant and, if lack of resources prevents them from following accepted standards of good practice, whether management rather than the clinician should be held to account. All of us involved in epilepsy care need to think about these issues because we are all potentially at risk, particularly because the United States 'no win no fee' system is developing here and litigants may be more prepared to 'try it on'.

The purpose of the symposium held during the Annual Scientific Meeting of the British Branch of the International League Against Epilepsy in Edinburgh in April 2000 was to examine some of the present standards of epilepsy management as reflected in the po-

tential outcome of litigation and in published guidelines. Participants in the meeting were given a series of epilepsy case histories to read before the meeting (and to bring with them). All these cases had been the subject of actual or threatened litigation and participants were asked to put themselves in the position of having just been in receipt of a solicitor's letter alleging negligence. The cases were discussed by a panel of clinicians (consultants, GPs and nurses) and a barrister. Since it is an urban medical myth that what happens in the US today will happen in the UK next week, there was also an American clinician as a discussant. There was an interactive voting system present in order that audience opinion and knowledge could be tested.

It should be remembered, in assessing the results of the interactive voting, that the audience (over 400) contained specialists in epilepsy care, neurologists, general physicians, GPs, nurse specialists and some medical students from whom different standards of knowledge might have been expected.

Note that in the following the case details have been altered in such a way as to preserve the anonymity of the patients without effecting the truth of the history.

CASE ONE. SUDDEN DEATH IN EPILEPSY (SUDEP)

Case history

Jane Hanna (Director of Epilepsy Bereaved) presented the first case history. A young man with a previous diagnosis of 'pseudoseizures' (jerking down the right side when walking) was referred to an epilepsy consultant. The patient had mild right-side weakness following a cerebral birth injury. Investigation showed that his right-sided seizures were actually movement-induced tonic–clonic seizures and that he was also experiencing secondarily generalized tonic–clonic seizures in his sleep. The seizures were unresponsive to conventional medication but the patient rejected trying new treatment (this decision was not recorded in the notes). A year later the patient died in his sleep of presumed sudden death in epilepsy (SUDEP). The consultant was invited to attend the inquest as a witness to fact.

Further developments

At the inquest, where the consultant was not legally represented, the patient's parents appeared (whom the consultant had never met). They were legally represented, and during an angry exchange announced that they would sue the consultant for not telling them that their son could die of his seizures. They alleged that an epilepsy advice organization had told them that parents should always be told of the risk of SUDEP. The following day a local newspaper, naming the consultant, ran the headline 'Doctor rapped for not warning of death risk'. The consultant chose not to respond to the newspaper headline and has heard no more from the parents.

The audience were asked the questions given in Table 1 at this point and their responses were recorded.

Discussion

Jane Hanna opined that when considering whether patients should be warned about SUDEP, an initial point to be raised is whether physicians should be doing this, bearing in mind that many are not well informed about the disorder. The starting point is therefore to ensure that health professionals themselves are fully informed. Secondly, we should think about this issue in terms of the ability of patients to participate in decisions that may have a direct impact on their lives. Certainly, in other countries (including Canada and Australia) the law has developed with regard to information disclosure and states that the ultimate choice of decision making should be for the patient. Patients need to have information that can help them achieve a balanced view. It might also be argued that if the family/carer have information about the fatality risks of seizures, they could employ appropriate first aid/resuscitation procedures. Although we do not have evidence suggesting that such procedures prevent SUDEP, it may be important that people do not feel an opportunity to try has been lost. (Some doctors, however, feel that to warn about the (comparatively low) risk of SUDEP will increase overprotection). Initially, when asked, the majority of the audience felt that all patients should be told of the risk of SUDEP (Table 1 question (A)). There was a fairly even split between those who would tell only the patient (45.2%) and those who would also warn the parents (52.7%), assuming the patient to be over 16 years old and of normal intelligence (Table 1 question (B)). After the case history had been presented, most audience members felt that they would seek legal advice if invited to attend an inquest (Table 1 question (C)). Dr Bill Smith (medical legal advisor) strongly advocated seeking such advice from a medical defence society. Although a significant proportion of the audience felt that they would seek advice from their Trust, it should be remembered that Trusts may have their own agenda.

Coroners are not permitted to place blame during an inquest, so the 'rapping' in this case would have originated from the family and the newspaper. Taking a considerate approach with relatives is particularly

Table 1:

(A)	Whom do you tell about the risk of SUDEP:	
	All patients	65.4%
	New patients	10.3%
	Those who do not comply with medication	22.1%
	No patients	2.2%
(B)	Assuming over 16 and normal intelligence: would you tell of the risk of SUDEP to:	
	Patient only	45.2%
	Parents only	1.4%
	Both	52.7%
	Neither	0.7%
(C)	If invited to attend an inquest would you seek legal representation:	
	Always (cannot be too careful)	31.9%
	Never (makes you look guilty)	1.4%
	Take advice from hospital Trust	31.2%
	Ask my Defence Society	35.5%

important. Focus groups with relatives bereaved by SUDEP have shown that what families actually want after the death, is to have an invitation from those involved in the patient's care to discuss what happened. Frequently, their aim is to ensure that care is good for other patients.

Following the discussion, opinion on who should be warned of SUDEP had changed, with 53.3% of those present starting that they would now warn more patients about the condition.

CASE TWO. INCORRECT DIAGNOSIS?

Case history

Professor Richard Dasheiff (an epileptologist from Oklahoma) described the case history of a 7-year-old child who developed drop attacks. During the attacks, the child fell suddenly and floppily to the floor, often hitting her head. She was pale and inert but not stiff and did not jerk. She recovered quickly after these attacks with few post-ictal symptoms.

Electroencephalogram (EEG) examination revealed 'excessive slow activity' in the right-fronto-temporal region immediately after a seizure, and a clinical diagnosis of atonic seizures was made. Many treatments were tried but only phenytoin had a beneficial effect on the condition. However, this was eventually withdrawn due to unacceptable side effects. At the age of 18, having received a poor education through frequently missing school, she was transferred to adult neurological care. An EEG and magnetic resonance imaging scan (MRI) were both normal but no attempt to monitor the seizures was made as it was felt that they were not occurring frequently enough (once or twice a week). She failed to gain employment and lived quietly at home with her ageing parents.

At this stage the audiences opinion was taken on the points given in Table 2.

Table 2:

(A)	On the facts presented what investigation would you most like to do next?	
	Ambulatory EEG	24.0%
	Video/EEG monitoring	57.5%
	Sleep deprived EEG	5.5%
	ECG	13.0%
(B)	Do you have your own ideas about the diagnosis on the facts presented?	
	Emotional pseudoseizures	10.9%
	Resistant frontal epilepsy	37.0%
	Resistant primary generalized epilepsy	10.9%
	Syncope	41.2%

Further developments

At the age of 27 the patient fell and fractured her shoulder. When admitted to hospital an electrocardiogram (ECG) showed a corrected QT interval of 670 milliseconds. She was admitted to a cardiology unit where a pacemaker was fitted which prevented further drop attacks. The consultant neurologist, the GP and the paediatrician received a solicitor's letter alleging the diagnosis of the Romano Ward syndrome (prolonged QT interval) should have been made earlier. The letter stated that not to perform an ECG and video monitor seizures in a person with apparently resistant epilepsy was negligent. Substantial damages were claimed for the patient's poor education and consequently poor employment prospects. In court a renowned, but long-retired, neurologist gave his expert opinion that a routine ECG was not necessary for the investigation of epilepsy, and the case was settled out of court.

Discussion

When a patient is not responding to treatment as expected, ideally they should be moved to a more advanced level of care. However, circumstances do not always permit this and the care that the patient in this case received can be considered as standard. Since doctors practice to a medical standard, not a legal one, a misdiagnosis does not equate with negligence. The Romano Ward syndrome was not widely reported in the literature until 1999, and even then was not commonly mentioned in standard epilepsy textbooks. From a legal perspective, it is important to consider what constitutes an expert opinion. As discussed earlier, it is preferable to have an expert witness who has specialist epilepsy knowledge. The evidence base from which such experts draw their opinions should also be taken into account.

The original diagnosis in this case appears to have been based on ambiguous EEG findings. EEGs are difficult to interpret in children and might have been misleading if they were recorded immediately after the patient had struck her head. Had there been more focus on the simple question 'Is this epilepsy or not?', an ECG would have been performed. Moreover, the perception that this patient's epilepsy was well controlled was a confounding factor: had there been recognition that one or two seizures weekly is still a problem, re-investigation would have been carried out earlier.

After the initial symptoms the girl was experiencing had been presented, the majority of the audience (57.5%) felt that video EEG monitoring was the investigation they would most like to do next (Table 2 question (A)). The most likely diagnoses at this stage were thought to be frontal epilepsy resistant to medication (37.0%) or syncope (41.2%) (Table 2 question (B)). Interestingly, the majority of audience members when asked for a show of hands were of the opinion that it is mandatory to perform an ECG examination on a patient suspected of having epilepsy, unlike the expert neurologist in the case. It should be remembered that although a single ECG channel is usually recorded during video EEG monitoring, and can be obtained during ambulatory EEG monitoring, it may not be of sufficient quality to measure the PR or QT interval: proper measurement is obtained from a proper ECG. EEG monitoring might have revealed that the attacks were not epileptic, but may well not have revealed the true cause of them.

CASE THREE. INCORRECT INVESTIGATION?

Case history

Dr Colin Mumford (consultant neurologist) introduced the case history of a 38-year-old man who developed complex partial seizures in 1994. The patient suffered loss of awareness, left-sided motor phenomena and subsequent Todds paralysis. A General Physician investigated his condition by EEG and computed tomography (CT) scan, both of which were normal. An MRI scan was not performed as there was a 12 month waiting list for this investigation at the nearest regional centre (40 miles away) and referral could only be made to it by a neurologist: there was a 12 month wait for this opinion. The patient's seizures were controlled for 2 years with carbamazepine but returned in 1996 and became secondarily generalized. Re-investigation (using MRI) by another consultant (the patient had moved) revealed a large right-sided parietal astrocytoma which was judged to be inoperable: despite decompression and radiotherapy, the patient died.

Further developments

The original physician received a letter from a solicitor acting on behalf of the patient's family, alleging negligence in not detecting the tumour earlier when it might have been operable. The letter also alleged that examination by CT is known to be inferior to MRI in investigating epilepsy and that published guidelines compel the investigation of epilepsy by MRI. The case was settled out of court by the consultant's Trust without admission of liability, having obtained an expert opinion that the case was indefensible. This was despite the opinion of another expert (a neurosurgeon) stating that even if the tumour had been detected in 1994, it probably would not have been operable.

Discussion

It is clear from this case that the Trust had its own agenda, which was to avoid publicizing the fact that it was not delivering adequate care to patients. Consequently the matter was settled out of court.

Generally, MRI is thought to be a superior scanning technique compared with CT. However, it is not an ideal world and availability of MRI is often restricted. Furthermore, despite the accepted superiority of MRI, it may not always be the most appropriate imaging technique to use at the start of epilepsy. A controlled trial of the usefulness of CT vs. MRI at diagnosis would be invaluable. Although the solicitor in this case alleged that guidelines compel the use of MRI, in reality the wording of the guidelines is not that extreme. The guidelines of the Royal College of Physicians in England do suggest that MRI is appropriate in patients with localization-related seizures[3]. However, whilst suggesting that imaging be carried out in patients with localization seizures, the Scottish Intercollegiate Guidelines Network (SIGN) (`www.show.scot.nhs.uk/sign/`) acknowledges that local circumstances may require this to be a CT scan rather than MRI. GPs are in a difficult position as the 'gatekeepers' of resources. In fact, given that availability of MRI is a resource problem, it might be appropriate to sue the Trust involved for not providing the necessary resources.

Before the outcome of the case was discussed, the audience was divided on whether they would investigate all suspected epilepsy cases at onset by MRI scanning or only use it when there was clear clinical (EEG) evidence of partial onset (Table 3(A), question 3). At this stage is was felt the fault lay with the original physician for not pushing investigations and with the Trust for not improving facilities (Table 3(B),

question 3). Although some audience members were happy with their current scanning policy, discussing this case left a large number feeling that they would be more insistent with their Trust over providing improved scanning resources (Table 3(C), question 3).

CASE FOUR. LATROGENIC BRAIN DAMAGE?

Case history

Dr Greg Rogers (GP) reported the case history of a patient with a troubled background. The boy had a history of repeated short-term psychiatric care for overdosing and recurrent wrist slashing and developed seizures at the age of 14 following a sexual assault whilst in foster care. On investigation by a paediatrician, EEG examination revealed bilateral paroxysmal theta activity, although a CT scan was normal and drug treatment was started. At the age of 15 the patient was transferred to an adult neurologist, who continued to prescribe antiepileptic drugs (AEDs): a combination of sodium valproate and phenytoin provided the best control. He was reviewed yearly in the neurology clinic. Prescribing was carried out by the GP who measured his serum phenytoin levels every 6 months: levels were consistently reported as being in the upper part of the therapeutic range. At the age of 22, the patient complained of unsteadiness and nausea. He was re-referred to the neurologist who found his phenytoin level to be within the normal therapeutic range and ascribed his ataxic symptoms to 'attention seeking'. The patient's symptoms worsened and he was referred to the local psychiatric services: by this time he was wheelchair bound.

Table 3:

(A)	When should, in your opinion, epilepsy be investigated by MRI scanning?	
	All cases at onset	41.2%
	Evidence of partial onset	36.0%
	Cases with no response to treatment	19.1%
	If CT scan is normal	3.7%
(B)	Whom do you think is most liable for not investigating the patient fully?	
	The GP, for not insisting on better facilities?	33.6%
	His Trust, for not providing them?	25.8%
	The Regional Centre, for its obstructive policy?	18.0%
	None of them?	22.7%
(C)	As a result of this discussion will you:	
	Review your scanning policy?	26.6%
	Remain satisfied with you present policy?	21.8%
	Bully your Trust to improve?	42.7%
	Consult my Defence Society?	8.9%

Table 4:

(A)	At this stage what do you judge to be most likely to be wrong with the patient?	
	Conversion disorder	16.1%
	Pseudoseizures	50.0%
	Cerebellar ataxia	29.8%
	Attention seeking	4.1%
(B)	Is there an interaction between valproate and phenytoin?	
	No	14.5%
	Phenytoin increases the bound fraction of valproate	17.1%
	Both drugs compete for folic acid binding sites in the cerebellum	26.3%
	*Valproate increases the unbound fraction of phenytoin	42.1%
	*The correct answer	
(C)	Be honest—did you know of this interaction?	
	Yes	16.3%
	Vaguely	46.3%
	Not at all	36.6%
	Are you sure there is one—it's not in the BNF	0.8%
(D)	The remedy is:	
	Measure free phenytoin levels	12.3%
	Increase the dose of valproate	2.6%
	Add folic acid	2.6%
	Avoid this drug combination wherever possible	82.5%

At this point the audience were asked to indicate what they felt the diagnosis to be: most chose a psychological cause (Table 4 question (A)) They were also asked if they knew of any interaction between valproate and phenytoin (Table 4 question (B)), but less than 50% knew the right answer.

Further developments

The psychiatrist, suspecting that the patient's severe ataxia was organic, referred him to a second neurologist who agreed. An MRI scan showed severe cerebellar atrophy consistent with phenytoin intoxication. The GP received a solicitor's letter alleging negligence on the grounds that he prescribed the phenytoin and that he 'should have known of the dangers of prescribing phenytoin in patients taking valproate when the unbound fraction of phenytoin increases and that, in those circumstances, intoxication and cerebellar damage can occur even when serum levels of phenytoin (which measure both the bound and unbound fractions of phenytoin) appear to be within the therapeutic range'.

Despite an expert witness for the defence stating that he would not expect a GP to know of the interaction between phenytoin and valproate (since it is not mentioned in the British National Formulary or the standard British Textbook[4]), a second expert witness (a GP) disagreed. The case was settled against the GP for a large sum of money: the original neurology consultant was not involved in the dispute, although he ad-

mitted that he was not aware of the interaction himself. The patient, who clearly had a conversion disorder and not epilepsy, was taken off all AEDs but made only a partial recovery from his ataxia.

Discussion

A serum phenytoin level is a measure of the total phenytoin of which around 10% is free, unbound and active. Valproate co-therapy increases this unbound fraction so that although serum levels may appear normal, the proportion of active phenytoin may have reached damaging levels. However, this interaction is not widely reported in the British literature. If consultant neurologists should certainly be aware of it, what about GPs? Again, we come back to the question of what is a responsible expert opinion. Although the GP treating the patient was not aware of the interaction, he was correct in referring the patient to a neurologist when his condition changed. He also maintained good links between primary and secondary care. It should be remembered that GPs are dealing with an ever increasing number of drugs and that even with the aid of a computerized prescribing system, it is extremely difficult to stay informed of all possible interactions: the valproate/phenytoin is not to be found on British GPs computerized prescribing information.

Less than 50% of audience members identified correctly that valproate increases the unbound fraction of phenytoin (Table 4 question (B)). Almost half admitted when challenged (Table 4 question (C)) that they had previously been only vaguely aware of this interaction. The majority of those present (82.5%) stated that they would now avoid this drug combination wherever possible when asked what they would do in the future now they were informed of the interaction (Table 4 question (D)): this may be a slight over-reaction.

I was pleased to see that my little textbook[5] does warn against this little known but important interaction: since it is difficult to routinely measure unbound fractions of AEDs (a similar difficulty occurs in pregnancy) perhaps it is better to avoid the combination, although the interaction does not occur in every patient taking a mixture of the two drugs[6].

CASE FIVE. UNEXPECTED PREGNANCY?

Case history

Annette Russell (Nursing Development Manager) related the case of a 15-year-old girl with complex partial seizures. She had been under the care of the same paediatrician for 5 years and her seizures were well controlled with carbamazepine. The paediatrician had not enquired if she was sexually active when he arranged her transfer to an adult service, but on her last visit (to his clinic nurse) it was discovered she had been sexually active for some time. The nurse advised her to go to a family planning clinic but did not warn her that there was an interaction between carbamazepine and oral contraceptives as she assumed the clinic would do this. The family planning clinic prescribed an ordinary dose of the pill. When the patient arrived at the adult neurology clinic, examination revealed that she was 4 months pregnant: she decided to keep the child and was consequently forced to give up college.

Further developments

The paediatrician received a solicitor's letter alleging negligence in not informing the girl of the risk of pregnancy if the contraceptive pill is taken in combination with an enzyme-inducing AED. The letter also stated that it was a well know principle that doctors are responsible for the 'acts and omissions of their servants', in this case, the nurse. The defence of the paediatrician and his nurse was successful but the case is still being pursued against the doctor at the family planning clinic. Examination of the family planning clinic's records suggests that the girl did not inform the doctor that she was taking carbamazepine.

Discussion

From a legal standpoint it is the Trust who is responsible, not the individuals concerned. However, it is unfortunate that the nurse missed a valuable opportunity to warn the girl of the risk of interaction between carbamazepine and enzyme-inducing AEDs. It may also be considered as her responsibility to do so, given that she discovered the girl was sexually active. It was dangerous to assume that someone else would carry out this task.

Paediatricians may find cases such as this one particularly difficult to manage. Treating patients from a very early age may increase the difficulty of defining the point at which adult (sexual) issues should be discussed, particularly if the parents are present. The situation is further complicated by the fact that patients seeking contraception may prefer to visit a family planning clinic because they want to avoid their regular physician. Although this kind of independent advice is an important option for patients to have, it may remove any opportunity for further communication.

Table 5:

(A)	Which of these drugs is enzyme inducing as far as a combined oral contraceptive is concerned?	
	Gabapentin	19.0%
	Tiagabine	10.0%
	Topiramate	41.0%
	None of them	30.0%
	The correct answer is topiramate	
(B)	Which of these drugs is not enzyme inducing as far as a combined oral contraceptive is concerned?	
	Topiramate	15.3%
	Ethosuximide	48.0%
	Oxcarbazepine	19.4%
	All of them	17.3%
	The correct answer is ethosuximide	

The audience showed poor awareness of which drugs are enzyme inducing with respect to the contraceptive pill (Table 5 questions (A) & (B)), but were better than a recent survey of American neurologists[7]. The majority (73.2%) agreed that all female patients of child-bearing age should be asked if they are (or might become) sexually active before being prescribed an AED. The majority of the audience (78.9%) also felt that doctors should routinely ask women with epilepsy about their contraceptive practice.

CASE SIX. INCORRECT RISK ASSESSMENT?

Case history

Dr Bill Smith (medical legal advisor) presented the case history of a 20-year-old woman with juvenile myoclonic epilepsy seeking pre-conception advice, which, in her clinic, was delegated by the neurologist to a nurse. Her condition had been well controlled since the age of 14 with valproate (400 mg daily). The specialist epilepsy nurse informed the patient, when she asked for pre-conception advice, that the risk of foetal damage with this dose of valproate was low and advised that she should continue taking it. The nurse also advised that it was not clinic policy to prescribe a high dose of folic acid as there was no clear evidence to suggest that it protected against AED-induced teratogenesis and advised that she should continue to take the normal dose for all pre-conceptual women of 400 mcg daily.

At this stage a majority of the audience correctly identified that a family history was an important part of the woman's investigations (Table 6 question (A)).

Further developments

The nurse unfortunately did not ask whether there was a history of spina bifida in the patient's family. In fact, there was a history of the disease in the blood lines of both the patient and her partner and it was later discovered that the patient herself suffered from spina bifida occulta. The patient later conceived: the foetus was found to have a severe spina bifida and the pregnancy was subsequently terminated, with much emotional trauma to the woman and her partner.

The consultant received a solicitor's letter alleging negligence in not fully warning against the risks of spina bifida and stating that guidelines recommend a higher dose of folic acid. He was also held accountable for the 'acts and omissions' of his nurse. The case was considered indefensible. Although there is no evidence to support the benefit of high dose folic acid against AED-induced malformation, there is substantial evidence that it protects against the genetically determined risk of spina bifida. The claim was settled out of court by the employing Trust, but the consultant was held partly to blame for not ensuring that the person to whom he delegated the task of counselling was competent to do so.

Table 6:

(A)	Before assigning risk is there any other investigation you would like to carry out?	
	Blood level of valproate	17.0%
	Folic acid level	13.6%
	X-ray of lumbar spine	4.5%
	Family history	64.8%
(B)	In the human is there any published evidence to suggest that folic acid given to women taking valproate:	
	Protects against spina bifida	56.4%
	Does not protect against spina bifida	1.7%
	Increases the risk of spina bifida	3.4%
	*No evidence either way	38.5%
	*The correct answer	
(C)	In the *rat* is there any published evidence to suggest that folic acid given to females also taking valproate:	
	Protects against spina bifida	71.2%
	Does not protect against spina bifida	7.7%
	!Increases the risk of spina bifida	2.9%
	*No evidence either way	18.3%
	!This is true for the mouse exposed to phenytoin and folic acid pre-conceptually (Schardein *et al.*[8])	
	*The correct answer (Craig *et al.* 1999[9])	
(D)	In the light of the discussion would you give pre-conception advice to women with epilepsy:	
	Yourself	28.9%
	With a colleague	22.8%
	Would tell GP to do it	4.4%
	*Would refer to specialist clinic	43.9%
	*Very few such clinics exist	

Discussion

The General Medical Council has made it clear that duty can only be delegated to people who are capable of providing the standard of care that would be expected of (and by) the doctor. In this case, the nurse failed to take a full history having had that responsibility delegated to her. The British National Formulary (BNF) states the position on folic acid in detail. In cases where there is a family history of spina bifida, it advises a folic acid supplement of 5 mg before and during pregnancy, particularly during the first trimester.

Most of the audience agreed that they would have taken a family history in this case. The majority (56.4%)—(Table 6 question (B))—thought they were aware of evidence supporting the protective effect against spina bifida of folic acid in pregnant women taking valproate (although there is none). However, they were mainly unaware (Table 6 question (C)) that there is now evidence that a spina bifida pregnancy has occurred in a woman taking valproate even though she had been taking the higher dose of folic acid[9]. In light of the discussion, the majority of those present would either give pre-conception advice themselves or refer patients for specialist pre-conception advice (Table 6 question (D)). However, the problem in this case was mainly one of communication. If pre-conception advice is given by doctor and nurse together, a greater range of issues can be covered and any omissions identified (Fox, 2000)[10]. Use of a pre-conception counselling protocol is a valuable tool in this setting[11].

CONCLUSIONS

The content of this symposium was particularly topical. Following a recent Court of Appeal test case, and a consultation exercise by the Lord Chancellor, the National Health Service bill for clinical negligence looks set to rise again (Dyer, 2000)[12]. The Appeal Court's ruling states that damages compensating for the effects of injuries on bodily functions and enjoyment of life should increase by one third. This was actually a relief to the NHS: the court rejected a recommendation from the Law Commission for an increase of 50% to 100%.

The value of expert testimony and the relevance of clinical guidelines were among the important issues raised by our discussions. In several of the cases, the knowledge and motives of the expert witness were questionable at best. Stringent investigation of the background and level of knowledge of such a witness is called for: epilepsy specialists should volunteer their services as expert witnesses more frequently. Although guidelines can be introduced to a court by an expert witness as evidence of accepted standards of care, they cannot be substituted for expert testimony. However well-linked to evidence, clinical guidelines need to be interpreted and applied sensibly. There is no doubt that the widespread adoption of relevant evidence based guidelines cannot be seen as *prima facie* evidence of negligence.

It is important to bear in mind that the courts are only part of the system for resolving disputes. They are concerned with identifying where practices have fallen short, not necessarily in identifying good practice. Of course there are areas where the clinical management of epilepsy could be improved. The management of women with epilepsy has been the subject of recent attention and in response to this, effective guidelines have been produced (Crawford *et al.*, 1999)[13]. Another area of concern is delegation of duty, particularly as the numbers (and responsibilities) of specialist epilepsy nurses increase. The medico-legal issues surrounding epilepsy care are complex but more open debate will raise awareness of the main issues.

The meeting also demonstrated that knowledge in the epilepsy field is changing rapidly and the different practitioners who make up the 'epilepsy field force' do not share the same knowledge base and are sometimes surprisingly ignorant (for example, as to which AEDs are—or are not—enzyme inducing). We can do very little about the patchy nature of epilepsy services in the UK as individuals, but we can improve our knowledge and make sure that any advice we give (whatever our professional background) is informed up to date advice.

For this reason the Editor and Editorial Board of *Seizure* have decided, as a priority, to institute a Continuing Medical Education Section to enable our readers, whatever their discipline, to develop their knowledge and keep abreast of new developments in all fields of epilepsy. I am grateful to Professor Stephen Brown and his team for taking on this task and wish them all success.

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Tim Betts
Editor in Chief
President, British Branch of the
International League Against Epilepsy

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